Haematological Characterization among Heavy Alcohol Consumers in Osogbo Metropolis

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Abstract: Alcohol abuse is fast becoming a public health concern among the Nigerian youths and adults strata of the society. A study on the effects of heavy alcohol consumption on haematological parameters was conducted on 130 subjects, comprising of 46 non-alcohol consumers who served as controls and 84 heavy alcohol consumers to determine the possible haematological attendant risks of the social behavior. Their ages ranged between 18-60 years. Sysmex KX-2IN instrument was used to determine the haematological parameters such as PCV, Haemoglobin, Red blood cell, Platelets, White blood cells total and differential counts of neutrophil and lymphocytes automatically. The result shows a significant difference (p<0.05) in the values obtained for alcohol drinkers which are high compared to that of the control group which could be dehydration based haemoconcentration. This study showed that abstinence from alcohol consumption had no effects on haematological parameters while its heavy consumption has deleterious effects such as severe infections to consequences of bone marrow malfunctioning. Hence clinical history of alcoholism during haematological investigations could be helpful and recommended.

Key words: Alcohol, haematological parameters, infection and dehydration, blood cell, haemoglobin, Nigeria

INTRODUCTION

Heavy intake of alcohol is a leading cause of preventable mortality, second only to cigarette smoking in industrialized countries. Alcohol is implicated in >40% of all fatal traffic accidents in 25% of all general hospital admissions in liver and upper gastrointestinal cancers, suicides, sex crimes, industrial accidents, robbery and murder and foetal alcohol syndrome. In the context of promotion of disease or death there is evidence to suggest that moderate alcohol intake reduces risk of coronary heart disease, the leading cause of death in most affluent societies. Therefore, it is important to establish the benefits and harmful effect (s) of alcohol on specific cardiovascular disease outcomes (Oulett, 1979).

Acute and chronic alcohol abuses are common conditions in patients admitted to hospitals. Alcohol has widespread direct and indirect effects on the hematologic system which can mimic and or obscure other disorders. Leukocyte, erythrocyte and thrombocyte production and functions are affected directly. Liver damage secondary to alcohol abuse also impacts red blood cells and the hemostatic mechanisms. Nutritional deficiencies are caused not only by poor dietary habits practiced by alcohol abusers but by the effect of alcohol on the absorption, storage and utilization of several vitamins (Hermans, 1998).

Over the past 20-30 years, the medical position on alcohol has become some what more nuanced.

Investigations of large-scale observational cohorts have suggested that light to moderate drinking may be associated with decreased mortality rates and with decreased risk of cardiovascular disease (Lucas *et al.*, 2005). The subject of alcohol and heart attacks is important because the major cause of death in many countries is cardiovascular disease.

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Besides cardiovascular disease, many of the pathophysiological effects of alcohol ingestion are related to the pathway of ethanol metabolism (Peters and Preedy, 1998). Findings suggest that alcohol abuse results in diverse patterns of hematological effects and affects several cell lines. Alcohol suppress platelet production and causes thrombocytopenia which result in platelet abnormalities inhibition of platelet aggregation (Sellah and Bobzien, 1999).

However, some studies (Gronbaek *et al.*, 2000) but not all (Fuchs *et al.*, 1995; Rimm *et al.*, 1996) have suggested that wine may provide additional benefits over and above the effects of alcohol, perhaps because of antioxidant or other types of effects of non-alcohol grape chemicals like resveratrol (Stervbo *et al.*, 2007). Some investigators have been so intrigued by the possible benefits of low-dose alcohol that they have gone so far as to consider mechanisms by which alcohol can have salutary cardiovascular effects (Rimm *et al.*, 1996).

These include increased levels of high-density lipoprotein cholesterol and a decreased tendency to thrombosis. Combining these reported biological benefits with epidemiological findings, some medical organizations have stated that low levels of alcohol consumption maybe considered safe (Pearson, 1996) or may be a legitimate "item of discussion between physician and patient" (Goldberg *et al.*, 2001).

Unfortunately alcohol abuse is known to have a wide array of adverse effects on the transport medium in the cardiovascular system, blood. The mechanisms by which it does so have not yet been established but there is considerable evidence which reveals adverse effects on serum proteins, blood cells and their progenitors in the bone marrow (Jaana, 2004).

The impact of alcohol on the hematopoietic system can be divided into direct and indirect effects. Direct effects are primarily seen in the bone marrow and involve the white cell, red cell and platelet lines. Indirect effects are secondary to metabolic or physiologic alterations resulting in liver disease and to nutritional abnormalities, such as folate deficiency (Hermans, 1998).

Haematological abnormalities are frequently found in heavy-drinking alcoholics but anaemia is generally a rare complication. When present, haemolysis is considered to be one of the most common causes (Kristensson *et al.*, 2008).

Haematological components of blood are also valuable in monitoring feed toxicity especially with feed constituents that affects the formation of blood. A full haematological examination of the alcohol consumers is to include; Red cell indices; red blood cell count, packed cell volume, mean cell volume, mean cell haemoglobin concentration, white blood cell count, platelet count and cell morphology.

Cell counts reflect the kinetics of entry and loss of cells from circulation and cell morphology reflects the status of individual cells which is a direct reflection of the health of the bone marrow, the circulation and the tissues.

With the rising increase in the consumption of alcohol, a large number of scientific papers have being published concerning biological effects of alcohol consumption on the human body and from different part of the world but none has been reported in Osogbo.

In view of the dearth of information in this regard this research was therefore designed to evaluate the effect (s) of long term consumption of alcohol on cellular components of blood in alcohol addicts in Osogbo metropolis. This would be achieved by determining various haematological parameters in the study population.

MATERIALS AND METHODS

Subjects selection: About 130 individuals were involved in this study comprising of 84 habitual consumers of alcohol and 46 non-consumers that served as controls. Consumers of at least 4 bottles (of both beer and local gins)/day for a period of at least 5years were designated consumers and strictly non-alcoholic drinks consumers were designated as control. All subjects were apparently healthy without any form of disease and their age ranged within 19-50 years.

Research design: All the subjects were administered questionnaire to determine the amount of alcohol they consume daily and the duration after which their blood samples were taken. Informed consent was obtained from the suitable participants while unwilling consumers were excluded.

Sample collection and analysis: About 5.0 mL blood samples were collected into K_3 EDTA anticoagulant bottles and were analyzed automatically for Packed Cell Volume (PCV), Haemoglobin estimation, Red cell count, total White Cell Count (WBC), Platelet count, Mean Corpuscular Volume (MCV), Mean Corpuscular Haemoglobin (MCH) and Mean Corpuscular Haemoglobin Concentration (MCHC) using an autoanalyser (Sysmex KN-21N). The equipment is a three part differential haematological analyzer that processes approximately 60 samples h^{-1} and shows on the LCD

(Liquid Crystal Show) screen. The particle distribution curves of WBC, RBC and Platelets along with data of 19 other parameters were produced as the analysis result/sample.

Estimation haematological parameters and haematological indices: The methodology is by flow cytometry (direct current method) using suitable cell packs according to manufacturer's specification for the desired cell population using SYMEX KX-21N auto-analyzer machine instrument (Epson *et al.*, 2006).

Differential white cell count: Leishman staining technique was used according to Dacie *et al.* (2006).

Procedure: A thin film was made, dried and stained with Leishman stain. It was diluted with phosphate buffer at pH 7.4 Allowed to stain for 10 min and rinsed. The stained slide was allowed to dry and examined using battlement method with oil immersion objective (x100). 100 consecutive white blood cells were counted in all, indicating various types of leukocytes encountered recorded.

Statistical analysis: Student t-test and ANOVA were used to analyze the differences between the results.

RESULTS AND DISCUSSION

The results of the haematological parameters among the heavy alcohol consumers and the control subjects are as shown in Fig. 1, Table 1 and 2. The heavy alcohol consumers group records a significantly higher values ($p<0.05$) in most of the cellular constituents related blood parameters than the non-consumers.

Alcohol exerts a direct toxic effect to the bone marrow resulting in vacuolization of the bone marrow precursor cells, anaemia, leukaemia and thrombocytopenia. It also affects the function of the leukocytes and platelets. Haematological functions are affected indirectly from nutritional deficiency, chronic alcoholic liver disease and other metabolic derangement (Chu, 2000). This has also been supported by Hermans, 1998 who reported that alcohols have a wide spread direct and indirect effects on the haematological system which mimic and obscure other disorders. Leukocytes, erythrocytes and thrombocytes production and functions are affected directly. Liver damage secondary to alcohol abuse also impact on red blood cells and haemostatic mechanism. More importantly, not only by poor dietary habits practiced by alcohol abusers but also by the effects of alcohol on the consumption, storage and neutralization of several vitamins.
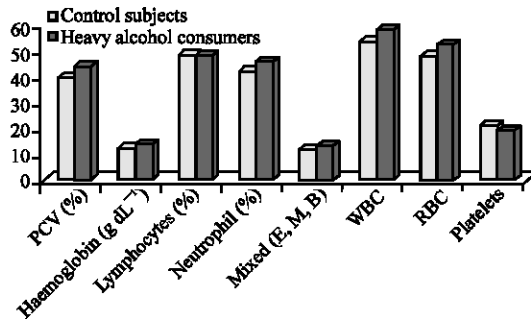

Fig. 1: Shows the distribution of haematological parameters among the studied population. A statistically significant increase was observed in packed cell volume and haemoglobin concentration ($p<0.05$) of the test group as compared with the control subjects, while there was no significant difference ($p>0.05$) in lymphocyte, neutrophil, mixed (Eosinophil, Monocyte, Basophil), white blood cell, red blood cell and platelet count in the studied population

The finding from this study has shown the effect of alcohol consumption on haematological parameters of some heavy alcohol consumers on Osogbo. The values obtained for haematological parameters in alcohol drinkers irrespective of sex showed a significant difference ($p<0.05$) in the haemoglobin concentration and packed cell volume which are higher when compared to those of the control this could be as a result of dehydration as reported by Kristensson *et al.* (2008).

This finding is comparable with previous research Shaper *et al.* (1985) and Oduola *et al.* (2005) who reported that alcohol causes highly significant positive association with globulin, potassium, haemoglobin, PCV, white cell count and calcium. All other haematological parameters in this research show no statistical significance difference ($p>0.05$) in both sexes of alcoholics when compared with the control group.

Although ethanol which is the basic constituent of alcohol has been reported to cause neutrophil and lymphocyte impairment which contribute to the increased frequency and severity of infections in alcoholics this present study showed no statistical significant difference ($p>0.05$) in the differential count between alcoholics and non-alcoholics.

There are differences in the effect of alcohol from one individual to another. When the beverage is sipped according to Brook and Brook (1975) the peak blood alcohol concentration is lower than when it is drunk. Luber *et al.* (1975) had also stated that it is about an hour or more after ingestion before alcohol in blood in the

Table 1: Shows the sex distribution of haematological parameters in the control and test population

Results	Sex	PCV (%)	Haemoglobin (g dL^{-1})	WBC (10^3 µL^{-1})	RBC ($\times 10^6$ µL^{-1})	Platelets (10^3 µL^{-1})	Lymphocyte (%)
Control	Female (n = 29)	37.652±3.523	11.890±1.240	5120.690±1316.483	4637.552±505.228	216068.970±74287.536	47.769±8.272
	Male (n = 17)	42.447±4.877	13.629±1.524	5758.824±1462.899	5054.706±587.834	208235.290±58217.834	49.006±6.544
	Total (n = 46)	39.424±4.512	12.533±1.582	5356.522±1391.506	4791.717±568.447	213173.910±68216.747	48.226±7.626
	P-value	<0.05	<0.05	>0.05	<0.05	>0.05	>0.05
Test	Female (n = 4)	36.800±0.455	11.725±0.263	5575.000±1345.053	4617.500±289.295	216750.000±48403.684	47.575±6.513
	Male (n = 80)	44.232±5.192	14.094±1.704	5769.000±1564.566	5153.775±709.029	188600.000±61133.512	48.000±9.203
	Total (n = 84)	43.879±5.311	13.981±1.738	5759.762±1548.230	5128.238±703.363	189940.480±60648.573	47.980±9.064
	P-value	<0.05	<0.05	>0.05	>0.05	>0.05	>0.05
Total	Female (n = 33)	37.548±3.11	11.870±1.164	5175.758±1307.199	4635.121±480.873	216151.520±71052.851	47.746±7.991
	Male (n = 97)	43.920±5.100	14.012±1.675	5767.216±1539.833	5136.412±687.549	192041.240±60800.376	48.176±8.774
	Total (n =130)	42.302±5.461	13.468±1.817	5617.077±1501.726	5009.162±676.108	198161.540±64142.985	48.067±8.554
	ANOVA	44.929	46.266	3.906	15.006	3.548	0.062
	P-value	<0.05	<0.05	>0.05	<0.05	>0.05	>0.05

Table 2: Shows the distribution of haematological parameters between the control male and test male subjects

Parameters	Subject	N	Mean±SD	T-test	p-value
PCV (%)	Control	17	42.447±4.488	1.316	<0.05
	Test	80	44.233±5.192		
Haemoglobin (g dL^{-1})	Control	17	13.629±14.094	1.038	<0.05
	Test	80	14.094±1.534		
WBC ($\times 10^3$ µL^{-1})	Control	17	5758.824±5769.000	0.025	>0.05
	Test	80	5869.000±1564.566		
RBC ($\times 10^6$ µL^{-1})	Control	17	5054.706±587.834	0.538	>0.05
	Test	80	5153.775±709.029		
Platelets ($\times 10^3$ µL^{-1})	Control	17	208235.290±58217.834	1.212	>0.05
	Test	80	188600.000±61133.512		
Lymphocyte (%)	Control	17	49.006±6.544	0.427	>0.05
	Test	80	48.000±9.203		
Neutrophil (%)	Control	17	40.106± 9.829	1.787	>0.05
	Test	80	45.651±11.953		
Mixed (Eosinophil+ Monocyte+Basophil) %	Control	17	13.550±3.142	0.105	>0.05
	Test	80	13.200±0.000		
	Control	17	49.006±6.544		

Table 2 shows a statistically significant increase in packed cell volume and haemoglobin concentration (p<0.05) in the male alcohol consumers than the control subjects, while there was no significant difference (p>0.05) in lymphocyte, neutrophil, mixed (Eosinophil, Monocyte, Basophil) white blood cell, red blood cell and platelet count between the control male and test male

blood stream reaches maximum concentration. As a result of the activity of the enzyme in metabolism, the percentage of alcohol to the blood is never constant over a longer period; it rises rapidly after consumption but then drops steadily when alcohol is ingested in combination with food.

CONCLUSION

In this study, alcohol intake has been shown to have serious effects on some haematological parameters which could predispose heavy alcohol consumers to infections and bone marrow malfunctions. Concerted effort is therefore required by public health educators to further inform the society of the haematological health risks of the social behavior. Also, clinical history of alcoholism during haematological investigation could also be helpful.

REFERENCES

Brook and Brook, 1975. Differences in hepatic and metabolic changes after acute and chronic alcohol consumption. Fed. Proc., 34: 2060-2074.

Chu, Y.C., 2000. Haematological effect of alcohol, long term ethanol consumption in alcoholics. Alcohol. Clin. Exp. Res., 24: 117-122.

Dacie, V.J., S.M. Lewis, B.J. Bain and I. Bates, 2006. Preparation and Staining Methods for Blood and Bone Marrow Films Practical Haematology. 10th Edn., Elsevier, India, pp: 70-71.

Epson, H.S., H.E.l.P. de Nemair and Terumo, 2006. Sysmex autoanalyzer model KX-21N. Sysmex Corporation, 1: 168-169.

Fuchs, C.S., M.J. Stampfer, G.A. Colditz, E.L. Giovannucci and J.E. Manson *et al.*, 1995. Alcohol consumption and mortality among women. N. Engl. J. Med., 332: 1245-1250.

Goldberg, I.J., L. Mosca, M.R. Piano and E.A. Fisher, 2001. AHA science advisory: Wine and your heart: A science advisory for healthcare professionals from the Nutrition Committee, Council on Epidemiology and Prevention and Council on Cardiovascular Nursing of the American Heart Association. Circulation, 103: 472-475.

Gronbaek, M., U. Becker and D. Johansen, 2000. Type of alcohol consumed and mortality from all causes, coronary heart disease and cancer. Ann. Intern. Med., 133: 411-419.

Hermans, E.H., 1998. Booze and blood: The effects of acute and chronic alcohol abuse on the hematopoietic system. J. Clin. Lab. Sci., 11: 229-232.

Jaana, L., 2004. Effect of alcohol consumption and acetyldehyde on blood cells and molecules. Pathogenic and Diagnostic Implications Acta, pp: 1-79.

Kristensson, A.A.A.S., S. Wallerstedt, C. Alling, G. Cederblad and B. Magnusson, 2008. Haematological findings in chronic alcoholics after heavy drinking with special reference to haemolysis. Eur. J. Clin. Invest., 16: 178-183.

Luber, H.L. Hoeksema and G.H. de Bock, 1975. The value of laboratory tests for the screening and recognition of alcohol abuse in primary care patients. J. Family Practise, 37: 268-276.

Lucas, D.L., R.A. Brown, M. Wassef and T.D. Giles, 2005. Alcohol and the cardiovascular system: Research challenges and opportunities. J. Am. Coll. Cardiol., 45: 1916-1924.

Oduola, T., O.G. Adeosun, T.A. Oduola, N.R. Agbaje and Z.A. Raheem, 2005. Biochemical and Haematological findings in alcohol consumers in ile ife, osun-state. Drinking Pattern, pp: 1-4.

Oulett, B.L., 1979. Alcohol and blood pressure. Am. J. Epidemiol., 109: 451-463.

Pearson, T.A., 1996. Alcohol and heart disease. Circulation, 94: 3023-3025.

Peters, T.J. and V.R. Preedy, 1998. Metabolic consequences of alcohol ingestion. Novartis Found Symp., 216: 19-24.

Rimm, E.B., A. Klatsky, D. Grobbee and M.J. Stampfer, 1996. Review of moderate alcohol consumption and reduced risk of coronary heart disease: Is the effect due to beer, wine, or spirits? BMJ, 312: 731-736.

Sellah, S. and W. Bobzien, 1999. Bleeding problem in patient with liver disease: Ways to manage the many hepatic effect of coagulation. Postgraduate Med., 106: 187-190.

Shaper, A.G., S.J. Pocock, D. Ashby, M. Walker and T.P. Whitehead, 1985. Biochemical and haematological response to alcohol intake. Ann. Clin. Biochem., 22: 50-60.

Stervbo, U., O. Vang and C. Bonnesen, 2007. A review of the content of the putative chemopreventive phytoalexin resveratrol in red wine. Food Chem., 101: 449-457.